Esophageal Hiatal Hernia

Some Aspects of Surgical Treatment

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• **Patients with esophageal hiatal hernia often have an array of distressing complaints and physical signs that are difficult to interpret. Physiologic and anatomic studies of the gastroesophageal area in the region of the esophageal hiatus of the diaphragm indicate the existence of a three-in-line sphincter group, consisting of the inferior esophageal constrictor, diaphragmatic pinchcock and cardioesophageal junction. These mechanisms, acting in unison, prevent regurgitation in normal persons.**

It also can be deduced from clinical, radiologic and experimental data that anatomic disturbances at the esophageal hiatus account for physiologic alterations. A reasonable explanation for the symptoms and signs of esophageal hiatal hernia can be made on the basis of the functional competence of the three-in-line sphincter mechanisms.

ALMOST ALL OBSERVERS agree that asymptomatic esophageal hiatal hernia needs no surgical treatment. Some disagreement on the importance of symptoms may exist between surgeons and internists. An internist's experience may include many mild cases while a surgeon's is often limited to patients with symptoms severe enough to warrant surgical intervention. Even in patients with hernias of considerable size one cannot predict that complications are likely. In recent years it has become recognized that size alone is an indefinite and unreliable criterion for surgical intervention. Not infrequently, small hernias may cause severe symptoms and oftentimes huge esophageal hiatal hernias may be discovered only during the study and treatment of other conditions (Figure 1 A, B).

The severity of symptoms is often unrelated to the degree of gastroesophageal reflux demonstrated in roentgenologic studies. Regurgitation of barium into the lower portion of the esophagus often occurs easily during the gastrointestinal study, yet the patient will have no symptoms of peptic regurgitation or esophagitis. Conversely, minimal gastric reflux may result in severe esophagitis. It is to be remembered, however, that such radiologic studies represent but a mere fraction of the total time that regurgitation may occur during the various phases of human activity. A barium study that shows no reflux does not necessarily indicate that regurgitation never occurs.

Nonsurgical treatment of symptomatic hiatal hernia has been described as being successful in 40 to 70 per cent of cases. Conservative therapy is designed to minimize gastroesophageal reflux and to dilute or neutralize the peptic activity of the regurgitant fluid. Treatment includes the regulation of diet, neutralization of gastric acidity, reduction of weight, and advice on the correct posture to assume while eating or sleeping. Eating while sitting rigidly upright often helps, as does raising the head of the patient's bed four to eight inches so that he may be in a semi-upright position while sleeping. The mere use of pillows to raise the patient's head, however, often defeats the purpose; pillows cause flexion of the body at the costal margins, bringing pressure by the anterior rib cage on the contents of the upper part of the abdomen, which in turn causes a pronounced increase in upper intra-abdominal pressure. If the patient is placed in a gradually declining position from head to feet, gravity can drain gastric juices away from the lower esophagus without alteration of the intra-abdominal pressure relationships (Figure 2).

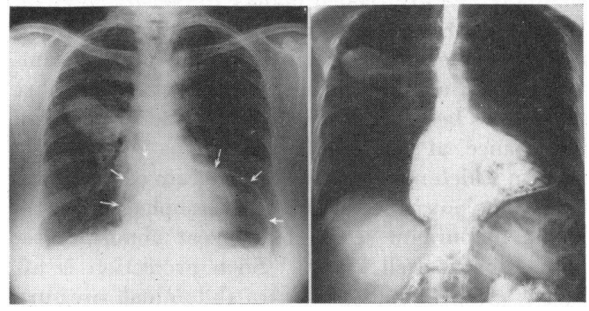

Figure 1.*—Completely asymptomatic large hiatal hernia, outlined by arrows in the plain film of the chest *(left)* and in a later film *(right)* by barium, in a patient being treated for a tuberculoma of the right upper lobe.

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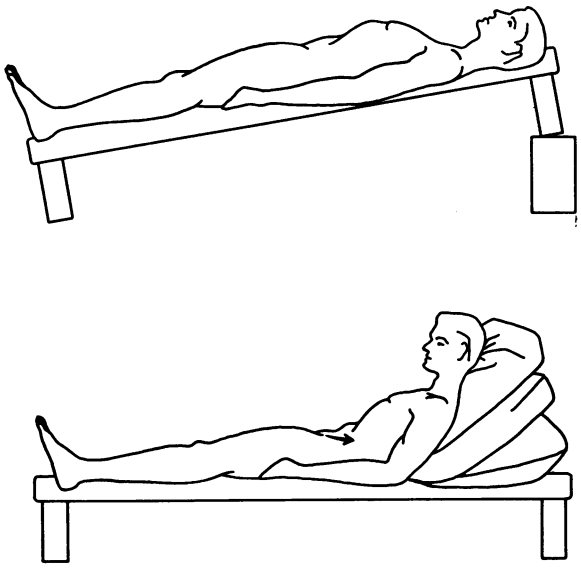

Figure 2.—A gradual decline from head to foot provides optimum gravity drainage. The use of pillows often promotes rather than prevents regurgitation into the lower esophagus.

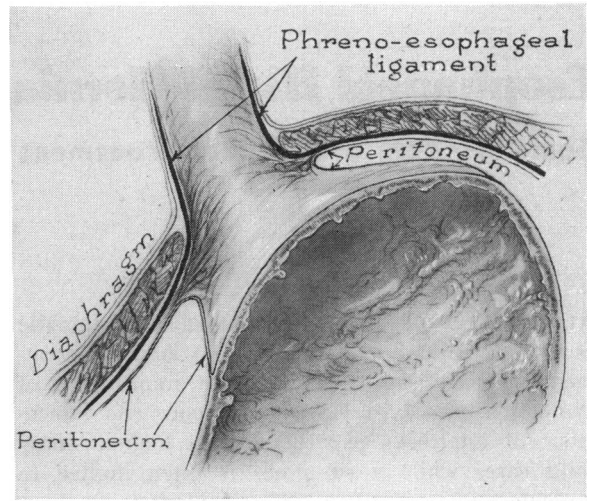

Figure 3.—The phrenoesophageal ligament consists of the fascia of the diaphragm which surrounds the esophagus at the hiatus and progresses along the lower esophageal segment to insert at the level of the inferior esophageal constrictor.

Conservative estimates indicate that at least 30 per cent of patients with symptomatic esophageal hiatal hernia will receive little or no benefit from conservative management. The reasons for failure are many and are related mainly to obesity, varying degrees of organic narrowing of the esophagus and a high degree of peptic activity. Probably most important is the unrelenting gastroesophageal reflux that occurs because of the anatomic and physiologic disturbance at the esophageal hiatus of the diaphragm which no conservative program can remedy.

That the normal structure of the esophageal hiatus of the diaphragm serves to prevent abnormal regurgitation is well known.[1] Such protection is afforded even though the intra-abdominal pressure exceeds that of the thorax by 4 to 12 millimeters of mercury. Even standing on one's head does not cause emptying of the stomach back into the esophagus. Furthermore, one can drink water in a normal fashion while in the head-down position without the occurrence of regurgitation. This suggests that although the cardioesophageal junction may open to allow passage of foods and fluids normally, the valvular mechanism prevents regurgitation even in these extreme circumstances. These simple physiologic facts indicate that a sphincteric mechanism not only exists but also functions dynamically to protect the esophagus as long as the region remains anatomically intact.

It is to be remembered that normally the terminal two to three centimeters of the esophagus lie below the level of the diaphragm and are exposed to positive intra-abdominal pressure. The combination of positive intra-abdominal tension and frequently an even greater positive intragastric pressure would force the contents of the stomach into the lower esophagus were it not for the existence of sphincteric action of some type at the gastroesophageal junction. This protective ability may be due to an intrinsic valve-like action at the cardioesophageal junction brought about by the interdigitation of the muscular coats of the esophagus and stomach as well as by the preservation of the angle at which the esophagus meets the stomach. The importance of this angle has been properly stressed.[2] It is conceivable, however, that the angle is important only insofar as it permits proper alignment and functioning of the interdigitating muscular fibers of each organ as they intertwine at the junction of the esophagus with the stomach. It is also possible that the angle of entry is important insofar as its presence indicates that the abdominal portion of the esophagus is of normal length and is correctly positioned and tethered.

The normal length of the esophagus may be a factor in the prevention of regurgitation, since the fundus of the stomach lying alongside the abdominal esophagus may by gaseous distention compress this portion of the esophagus against the left lobe of the liver. This, along with the gastroesophageal junction itself, may serve to prevent significant regurgitation yet allow enough physiologic relaxation to permit the orderly passage of food from the esophagus to the stomach in response to the peristaltic activity of the esophagus proximal to this area.

The competence of the cardioesophageal sphincter mechanism depends, therefore, upon normal anatomic relationships in and about the area of the esophageal hiatus of the diaphragm. These relation-

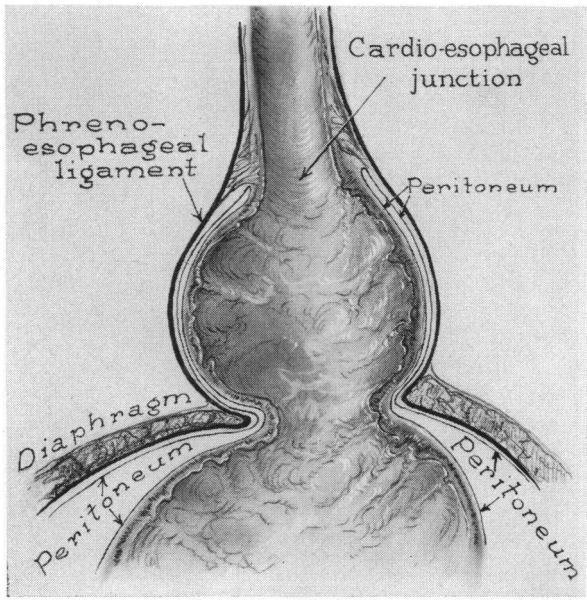

Figure 4.—Esophageal hiatal hernia of the sliding type. The phrenoesophageal ligament is elongated and weakened, allowing the lower esophagus and stomach to slide upward into the mediastinum. The normal angle of the junction of the esophagus with the stomach has been obliterated.

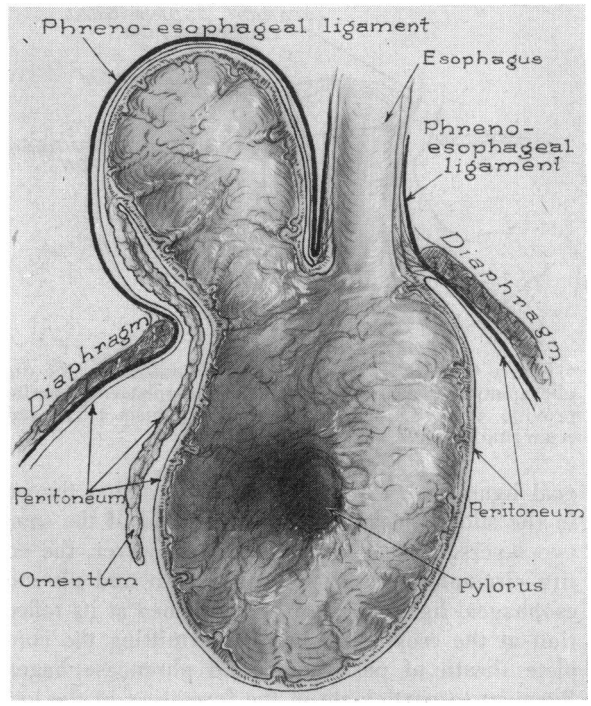

Figure 5.—Diagrammatic cross-section showing the normally placed cardioesophageal junction in the paraesophageal type of hiatal hernia. The herniated portion of the stomach lies alongside the lower esophagus.

ships are maintained by the integrity of the hiatus and particularly by the phrenoesophageal ligament which, when intact, restrains, along with the peritoneal reflections in this area, the lower end of the esophagus in its normal position below the diaphragm (Figure 3). As the esophageal hiatus becomes weakened and enlarged due to the patient's increasing age, obesity or other factors, the phrenoesophageal ligament must also be attentuated and weakened concomitantly. This sets the stage for further weakening and elongation of the ligament, aided by the upward force created by the differential positive pressure within the abdomen and the negative pressure within the thorax.

These alterations allow a loosening of the tethering effect upon the lower portion of the esophagus, permitting the entire segment, consisting of the lower esophagus and upper stomach along with the elongated phrenoesophageal ligament, to slide upward into the posterior mediastinum (Figure 4). This cephalad migration is enhanced by the natural elastic recoil of the esophagus which occurs during the act of swallowing. As a result a sliding hernia of the esophageal hiatus of the diaphragm occurs. In almost all instances the movement of these segments is a two-way transfer, since the shortening of the esophagus is physiologic rather than organic. The herniated viscera may reduce themselves into a normal state below the diaphragm when the patient is in an upright position, by the effect of gravity resulting from a full stomach after meals; or the reduction may be brought about by a definitive surgical procedure.[3]

As long as the lower esophagus, cardioesophageal junction and upper portion of the stomach remain displaced in the thorax, as in hiatal hernia of the sliding type, regurgitation leading to esophagitis is likely to occur since the area is completely disturbed anatomically and physiologically. In most instances, esophagitis causes the most significant symptomatology in patients with sliding hiatal hernias and is the main justification for surgical repair.

Hiatal hernia of the paraesophageal type presents quite a different problem with respect to etiology, symptoms and treatment. In herniation through the esophageal hiatus, the abdominal esophagus remains correctly positioned and maintains its normal relationship with the lesser curvature of the stomach. It follows, however, that since a part of the stomach herniates through the esophageal hiatus into the posterior mediastinum, a part of the circumferential span of the phrenoesophageal ligament must be carried upward into the thorax along with the similar and corresponding segment of the peritoneum that invests the stomach. It is possible that a segment of peritoneum is left behind in the thorax during embryonic development and constitutes the sac of a paraesophageal hernia. In either event the sac will be composed of two layers, namely the peritoneum and the elongated, weakened phrenoesopha-

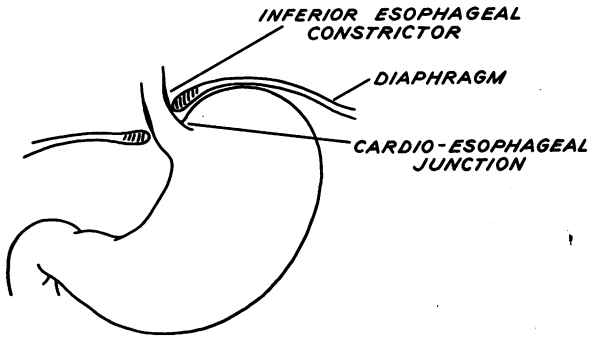

Figure 6.—The inferior esophageal constrictor, the diaphragmatic pinchcock and the cardioesophageal junction provide a three-in-line sphincter mechanism to prevent regurgitation.

geal ligament. It is to be remembered that the sac in the sliding hernia is also composed of the same two layers. In the latter instance, however, the entire circumference of the peritoneum and phrenoesophageal ligament becomes weakened at its reflection at the esophageal hiatus, permitting the complete sheath of peritoneum and phrenoesophageal ligament to participate in the formation of the hernia (Figure 5).

It is well accepted that a valve-like action exists at the cardioesophageal junction. The role of the two other actual or possible sphincter mechanisms acting in and upon the lower part of the esophagus is still not completely understood. These mechanisms are the so-called diaphragmatic pinchcock and inferior esophageal constrictor (Figure 6). The exact role of the diaphragm in the prevention of regurgitation is as yet undetermined and opinions vary considerably regarding its function, or lack of it. There is little doubt that in normal persons some active muscular contraction occurs at the esophageal hiatus of the diaphragm. This can be demonstrated easily if one inserts a finger into the lower esophagus during gastrostomy. The pressure exerted upon the finger by the diaphragm is easily recognizable. How much similar action is possible when the esophageal hiatus is weakened and enlarged can only be surmised. Spasm of the diaphragm is possible even with an enlarged hiatus and may even account for some of the confusing symptoms and signs associated with hiatal hernia.

Above the level of the diaphragm the third sphincteric mechanism (inferior esophageal constrictor)[4-7] may act along with the other two (diaphragmatic pinchcock and gastroesophageal junction) as a third-in-line mechanism to prevent regurgitation. That there is some sort of process which causes hesitation in the downward descent of a column of barium in the lower esophagus, two to four centimeters above the diaphragm, has been recognized for many years. Almost always the column will be delayed momentarily at this point before it passes into the remainder of the esophagus and thence into the stomach (Figure 7). That portion of the esophagus between the inferior constrictor and the gastroesophageal junction has been called the gastroesophageal vestibule; the area immediately above the constrictor has been termed the phrenic ampulla. It has been shown by balloon pressure studies that the peristaltic activity of the gastroesophageal vestibule is quite different from that of the remainder of the proximal esophagus.

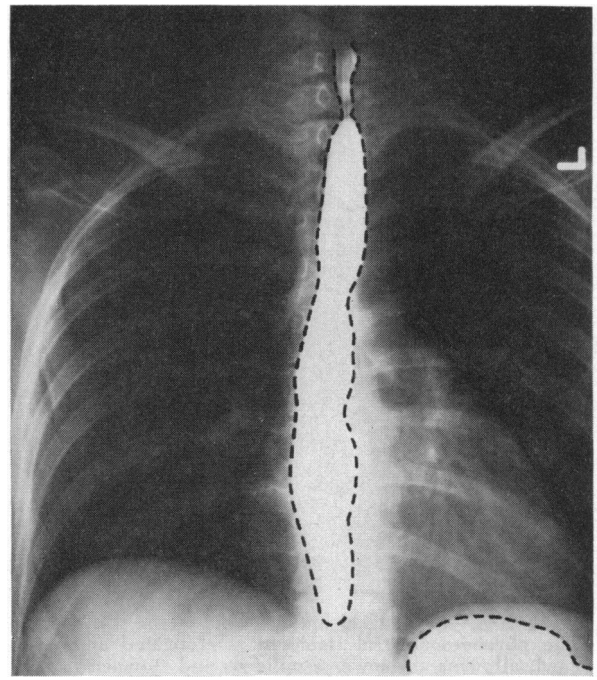

Figure 7.—The functional existence of a lower esophageal constrictor is demonstrated by the momentary hesitation of a column of barium in the lower esophageal region.

The phrenoesophageal ligament, as it reflects itself from the undersurface of the diaphragm upward to become the fascia of the esophagus, is finally inserted into the circular muscle of the esophagus at the level of the inferior esophageal constrictor. The ligament serves as a natural antagonist to the longitudinal pull of the esophagus. Some investigators have described an anatomic thickening of the circular muscle at the level of the constrictor; this constitutes anatomic evidence of a sphincter and augments the evidence gained from physiologic and radiologic observations.

The inferior esophageal constrictor may serve to prevent esophagitis in some instances and probably acts as part of a team consisting of the three-in-line sphincters which act in physiologic unison to allow antegrade passage of food and fluids into the stomach yet prevent significant regurgitation. The inferior constrictor, however, may be strong enough

to prevent regurgitation from a herniated gastric pouch, even when the other two sphincteric mechanisms may be non-functional. If so, this sphincteric action may explain the absence of demonstrable esophagitis by roentgenograms and esophagoscopy even when symptoms of substernal burning pain are present. In this situation the gastric juice is contained within the gastric pouch even though it is displaced into the mediastinum, thus providing the substernal burning pain suggestive of esophagitis but which in reality is due to gastritis in the herniated segment of the stomach (Figure 8). In these circumstances esophagoscopy will demonstrate a normal lower esophageal segment.

In hiatal hernia of the paraesophageal type, symptoms of esophagitis are the exception rather than the rule. This is true because the length of the esophagus and the relationships at its junction with the lesser curvature of the stomach are preserved physiologically and anatomically. Instead of the substernal or epigastric burning pain so often produced by esophagitis, patients with the paraesophageal hernia commonly complain of pressure symptoms. Palpitations are not infrequent and there is often a sense of fullness or actual pressure in the lower anterior thorax. It must be acknowledged that as a considerable portion of the stomach rolls upward into the mediastinum the gastroesophageal junction will occasionally be displaced by traction, allowing some regurgitation to occur. In such patients esophagitis and distressing pressure symptoms may coexist.

The hemorrhage from an esophageal hiatal hernia which occasionally occurs in a patient without esophagitis can also be explained on the basis of a gastric pouch lying above the diaphragm. Here again, if the inferior esophageal constrictor functions well the action of the gastric juice will be limited to the mucosa of the herniated stomach and may produce hemorrhage even though there is no esophagoscopic evidence of esophagitis. The hemorrhage may be promoted further by venous stasis of the herniated stomach. If both the diaphragmatic pinchcock and the inferior constrictor remain competent, a closed-pouch effect may occur at either end of the herniated portion of the stomach, enhancing the possibilities of gastritis and hemorrhage by means of stasis of gastric contents. If the action of the diaphragmatic pinchcock is excessive it may force peptic juice out of the herniated stomach into the lower esophagus, regardless of the strength of the inferior esophageal constrictor.

The operative repair of an esophageal hiatal hernia is also related to the concept of the three-in-line sphincter mechanisms as well as to the reconstitution of the normal anatomy of the region of the esophageal hiatus. In the operative repair, nothing can be done to the inferior esophageal constrictor except that in the surgical shortening of the phrenoesophageal ligament to which it is attached, the inferior constrictor may again become more competent. The diaphragmatic pinchcock action, if indeed it does exist, can be restored by approximating the crura posteriorly so that the hiatus again accommodates the esophagus.

THE DIAPHRAGMATIC PINCHCOCK AND THE INFERIOR ESOPHAGEAL CONSTRICTOR

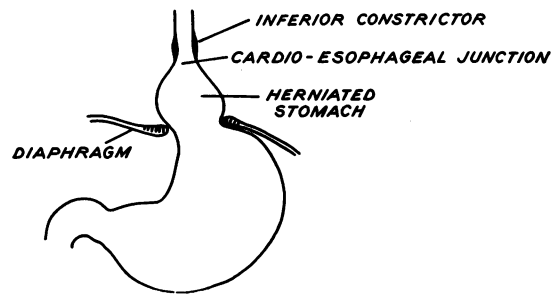

Figure 8.—A competent inferior esophageal constrictor may prevent esophagitis even in the presence of a sliding esophageal hiatal hernia.

By excision of the hernial sac and shortening the phrenoesophageal ligament, the physiologically shortened esophagus is again lengthened and brought down below the level of the diaphragm. As this is accomplished, the normal visceral relationships and physiologic activity are functionally restored. At least two of the three-in-line sphincter mechanisms are thus surgically restored; the sphincteric mechanism at the gastroesophageal junction is brought about by restoring the normal anatomic relationships and cardioesophageal angle and, secondly, closure of the crura snugly about the abdominal esophagus restores the diaphragmatic pinchcock. If in addition the inferior esophageal constrictor remains functional, then a complete restoration of the three-in-line mechanisms preventing regurgitation is established.

The concept of the three-in-line sphincter mechanisms, either acting in unison or one or more acting normally, may also account for some of the good results produced after various methods of surgical repair. One such procedure recommended by some surgeons, especially for elderly patients, is simple crushing of the left phrenic nerve. Good results can be expected from this procedure in patients in whom the diaphragmatic pinchcock is spastic, and in such cases paralysis of the phrenic nerve may prevent symptoms. Other surgeons have suggested transplantation of the esophageal hiatus to the dome of the diaphragm, especially when the esophagus has become moderately shortened. In this instance no

diaphragmatic pinchcock action is possible. Good results from such a procedure are dependent, therefore, on restoration of at least one, and perhaps two, of the other sphincter mechanisms.

Perhaps the large yet asymptomatic esophageal hiatal hernia can also be explained on the basis of the three-in-line sphincter mechanisms. In this instance one may visualize that the diaphragmatic pinchcock is completely functionless and without contractile power because of the huge size of the hiatus. However, a strong functioning inferior constrictor may serve to prevent regurgitation and the disabling effects of esophagitis. Why the patients do not have symptoms referable to the volume of the herniated stomach is difficult to explain but may be related to the slow development of the hernia and gradual accommodation of the herniated stomach within the thorax.

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